PB82-118126

# Effects of Hydrostatic Pressure on Steelhead Survival in Air-Supersaturated Water

(U.S.) Corvallis Environmental Research Lab., OR

1980

U.S. Department of Commerce
National Technical Information Service
NTIS

| TECHNICAL REPORT DATA<br>*(Please read Instructions on the reverse before completing)* | | |
|---|---|---|
| 1. REPORT NO.<br>EPA-600/J-80-349 | 2.<br>Journal article | 3. RE P882-118126 |
| 4. TITLE AND SUBTITLE<br>Effects of Hydrostatic Pressure on Steelhead Survival in Air-Supersaturated Water | | 5. REPORT DATE<br>October 1981<br>6. TION CODE<br>307081 |
| 7. AUTHOR(S)<br>M.D. Knittel, G.A. Chapman, R.R. Garton | | 8. PERFORMING ORGANIZATION REPORT NO. |
| 9. PERFORMING ORGANIZATION NAME AND ADDRESS<br>Environmental Research Laboratory<br>Office of Research and Development<br>U.S. Environmental Protection Agency<br>Corvallis, Oregon 97333 | | 10. PROGRAM ELEMENT NO.<br>11. CONTRACT/GRANT NO. |
| 12. SPONSORING AGENCY NAME AND ADDRESS<br>same | | 13. TYPE OF REPORT AND PERIOD COVERED<br>14. SPONSORING AGENCY CODE<br>EPA/600/02 |
| 15. SUPPLEMENTARY NOTES<br>Transactions of the American Fisheries Society 109:755-759, 1980 | | |

16. ABSTRACT

Juvenile steelheads (Salmo gairdneri) were placed in cages and suspended at various depths in water supersaturated with air at levels from 120 to 140% of normal atmospheric gas pressure. survival times of fish held at 10, 50, and 100 cm depth increased with increasing depth at a given level of supersaturation. When the hydrostatic pressure (7.4 mmHg per 10 cm of water depth) was subtracted from the excess gas pressure (relative to surface barometric pressure), mortality curves (times to 50 % mortality versus excess gas pressure), for fish at all three depths essentially coincided. The significant measure of supersaturation appears to be the pressure of dissolved gases in excess of the sum of barometric and hydrostatic pressures. Steelheads held near the surface in supersaturated water for a near-lethal period and then lowered to a depth providing total hydrostatic compensation appeared to recover completely in about 2 hours. The longer fish remained at depth, the longer their survival time when they subsequently were re-exposed to surface conditions.

| 17. KEY WORDS AND DOCUMENT ANALYSIS | | |
|---|---|---|
| a. DESCRIPTORS | b. IDENTIFIERS/OPEN ENDED TERMS | c. COSATI Field/Group |
| | | |

REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U.S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA 22161

| 18. DISTRIBUTION STATEMENT | 19. SECURITY CLASS *(This Report)* | 21. NO. OF PAGES |
|---|---|---|
| Release to public | Unclassified | |
| | 20. SECURITY CLASS *(This page)* Unclassified | 22. PRICE |

EPA Form 2220-1 (9-73)

EPA-600/J-80-349
Journal Article

*Transactions of the American Fisheries Society* 109:755–759, 1980

# Effects of Hydrostatic Pressure on Steelhead Survival in Air-Supersaturated Water

M. D. KNITTEL, G. A. CHAPMAN, AND R. R. GARTON

*Corvallis Environmental Research Laboratory, United States Environmental Protection Agency*
*1350 Southeast Goodnight Avenue, Corvallis, Oregon 97330*

## Abstract

Juvenile steelheads (*Salmo gairdneri*) were placed in cages and suspended at various depths in water supersaturated with air at levels from 120 to 140% of normal atmospheric gas pressure. Survival times of fish held at 10, 50, and 100 cm depth increased with increasing depth at a given level of supersaturation. When the hydrostatic pressure (7.4 mm Hg per 10 cm of water depth) was subtracted from the excess gas pressure (relative to surface barometric pressure), mortality curves (times to 50% mortality versus excess gas pressure) for fish at all three depths essentially coincided. The significant measure of supersaturation appears to be the pressure of dissolved gases in excess of the sum of barometric and hydrostatic pressures. Steelheads held near the surface in supersaturated water for a near-lethal period and then lowered to a depth providing total hydrostatic compensation appeared to recover completely in about 2 hours. The longer fish remained at depth, the longer their survival time when they subsequently were reexposed to surface conditions.

When air is dissolved in water at pressures exceeding one atmosphere, more gas is driven into solution than is normal for most surface waters; such waters are said to be supersaturated. In the absence of vigorous surface turbulence, reequilibration to the normal saturation level occurs slowly even in shallow water. Conditions conducive to the production of supersaturation can occur in plunge basins below the spillways of dams, in power plants where saturated cooling waters are warmed and gas solubility is reduced, and in penstocks or other water pipes where air and water can be mixed at high pressures.

Dissolved gas levels between 100 and 140% of normal saturation have been reported in natural waters as a result of these conditions. When aquatic animals, especially fish, are exposed to water containing gas levels over 110%, they are often killed by air emboli collecting in vital organs. Stroud et al. (1975) have described the pathology associated with exposure of fish to supersaturated water. Several comprehensive reviews of the causes and effects of supersaturation have been published (Rucker 1972; Wolke et al. 1974; Fickeisen and Schneider 1976; Weitkamp and Katz 1980).

Although the lethality of air-supersaturated water to salmonids has been well documented, most of the work has been done in shallow tanks where water depth is not a major factor affecting the response of fish to supersaturation. Greater water depth adds the variable of hydrostatic pressure which inhibits the formation of air emboli in tissue fluids supersaturated with gases relative to ambient atmospheric pressure.

Previous supersaturation studies have shown qualitatively that deep-water exposure or intermittent deep- and shallow-water exposures produce lower mortality than shallow-water exposure (Dawley et al. 1976; Weitkamp 1976). However, these studies allowed considerable variability of depth so that exposure levels could have varied considerably, making quantitative analysis of effects difficult. Fickeisen and Montgomery (1978) conducted a study similar to ours, but they used only one level of supersaturation and did not fully quantitate the effects of hydrostatic compensation.

The first objective of this study was to determine if compensating hydrostatic pressure and excess gas pressure are simple additive factors determining fish survival. The second objective was to determine the extent to which total hydrostatic compensation can reverse the effect of a previous near-lethal exposure to supersaturation.

## Methods

Juvenile steelheads (*Salmo gairdneri*) were reared at the Corvallis Environmental Research

TABLE 1.—*Temperature and nominal and measured total gas concentrations during supersaturation experiments with steelheads.*

| Test | Temperature C | % total gas saturation | | | |
|---|---|---|---|---|---|
| | | Nominal | Measured | | |
| | | | N | Mean[a] ± SD | Range |
| 1 | 10.8 | 140 | 27 | 139.8 ± 0.4 | 138.9–140.3 |
| 2 | 10.0 | 140 | 39 | 139.6 ± 0.9 | 135.5–141.0 |
| 3 | 11.0 | 135 | 41 | 136.4 ± 0.7 | 135.2–137.3 |
| 4 | 10.0 | 135 | 27 | 135.1 ± 0.2 | 134.8–135.7 |
| 5 | 11.5 | 130 | 36 | 130.0 ± 0.8 | 129.0–130.8 |
| 6 | 11.7 | 130 | 60 | 130.2 ± 0.6 | 128.1–130.9 |
| 7 | 11.0 | 130 | 55 | 130.4 ± 0.5 | 129.0–130.5 |
| 8 | 9.0 | 125 | 45 | 124.9 ± 0.4 | 124.0–125.7 |
| 9 | 9.5 | 125 | 25 | 125.0 ± 0.5 | 124.0–126.0 |
| 10 | 9.2 | 125 | 31 | 124.9 ± 0.6 | 124.0–125.2 |
| 11 | 10.2 | 120 | 26 | 120.1 ± 0.3 | 119.8–120.8 |
| 12 | 11.2 | 120 | 27 | 120.0 ± 0.4 | 119.4–120.8 |
| 13 | 9.5 | 120 | 41 | 120.2 ± 0.5 | 118.8–120.9 |
| 14[b] | 14.0 | 120 | 45 | 119.7 ± 0.4 | 118.9–120.5 |
| 15[b] | 12.0 | 120 | 54 | 119.8 ± 0.4 | 118.7–120.4 |
| 16[b] | 13.6 | 125 | 25 | 126.6 ± 0.5 | 125.2–127.0 |
| 17[b] | 14.0 | 125 | 42 | 126.5 ± 0.6 | 125.7–127.7 |
| 18[b] | 12.5 | 130 | 15 | 130.5 ± 0.4 | 130.3–131.3 |

[a] Means of measurements in four tanks per test.
[b] Variable-depth exposure tests.

TABLE 2.—*Calculated percentage reduction (compensation) in air supersaturation based on pressure of the water column.*[a]

| Water depth (cm) | Water column pressure (mm Hg) | Compensation (percentage of atmosphere) |
|---|---|---|
| 10 | 7.4 | 1.0 |
| 20 | 14.8 | 1.9 |
| 30 | 22.2 | 2.9 |
| 40 | 29.6 | 3.9 |
| 50 | 37.0 | 4.9 |
| 60 | 44.4 | 5.8 |
| 70 | 51.8 | 6.8 |
| 80 | 59.2 | 7.8 |
| 90 | 66.6 | 8.8 |
| 100 | 74.0 | 9.7 |
| 110 | 81.4 | 10.7 |
| 120 | 89.8 | 11.7 |
| 130 | 96.2 | 12.7 |

[a] % compensation = [Water depth (cm) × 0.740 (mm Hg/cm water)]100/760 (mm Hg).

Laboratory from eggs obtained from the North Fork Alsea Hatchery, Alsea, Oregon. Fish culture and test facilities were supplied with soft well water (Samuelson 1976). The fish were reared on Oregon Moist Pellet diet but were not fed during the tests.

Tests were conducted by holding fish in cylindrical cages, 71 cm in diameter and 20 cm deep, suspended at various depths in four fiberglass tanks 3.1 m high and 79 cm in diameter. The water supply to the tanks was supersaturated to desired levels by regulating the flow of compressed air into a pressure chamber (Nebeker et al. 1976) which supplied water to each tank at a rate of 14 liters/minute. Nominal total gas saturations ranged from 120 to 140% (Table 1).

The air supersaturation of water was measured with a Weiss saturometer (dissolved gas tension meter), which registered the difference between the dissolved gas and atmospheric pressures (Nebeker et al. 1976). The tension meter was calibrated periodically with a Van Slyke gas analyzer. All saturation levels are expressed as percent of saturation with respect to ambient atmospheric pressure at the water surface and were uniform throughout the water column in each tank. Calculation of gas saturation levels followed the conventional procedure of dividing dissolved gas pressure (less water vapor pressure) by barometric pressure and multiplying the quotient by 100 (Fickeisen et al. 1975).

Tests were begun by placing 20 fish in each cage and then lowering the cage to the desired depth. Periodic in situ observations were made for deaths and for the saturation level of air in the water. A fifth group of 20 fish was placed in a cage and held in shallow ambient well water as a control. The median survival time (LT50) for fish in each cage was calculated on the assumption of a log-normal relationship between mortality and exposure time.

### *Constant Depth Exposure*

The force driving the formation of gas emboli is the existence in body fluids of dissolved gas tensions in excess of the sum of atmospheric and hydrostatic pressures. In these experiments the excess gas tension was greatest in the cages just below the surface where the hydrostatic pressure at 10 cm was only about 7.4 mm Hg.[1] Because atmospheric pressure during the tests was always near 760 mm Hg, each percent supersaturation was equivalent to 7.6 mm Hg.

[1] $H_2O$ pressure (mm Hg)
$$= \frac{\text{mm } H_2O \text{ depth} \times \text{density } H_2O}{\text{density Hg}}$$
$$= \frac{\text{depth} \times 1.000 \text{ g/cm}^3}{13.596 \text{ g/cm}^3}.$$

Therefore, each 10 cm of water depth should have created pressure nearly sufficient to counteract the gas tension created by one percentage unit of supersaturation (0.01 atmosphere) (Table 2). If the time to death due to gas emboli formation is solely a function of the excess gas pressure in the body fluids, then survival times at 10, 50, and 100 cm depth should be equal at identical excess gas pressures after adjustment for depth compensation. This was tested by the procedure outlined above.

*Intermittent Depth Exposure*

Another potential effect of increasing depth is the resolubilizing of emboli formed at lower hydrostatic pressures. Thus, fish in which potentially lethal emboli have formed may survive if they enter deeper water. The time required for gas resorption is unknown as is the influence of an earlier surface exposure and recovery at depth on survival during a second surface exposure. To test these effects, fish near the surface were exposed to lethal levels of supersaturation for several hours, lowered to a compensating depth (3 m) for 1, 2, or 3 hours, and then returned to the surface.

Total gas saturation levels in these tests were 130, 125, and 120%, and the times of initial exposure were 2.3, 3.3, and 5.9 hours, respectively. These periods were previously determined to be just short of the time to first death at these saturation levels.

## Results and Discussion

When caged steelheads were exposed to a given level of gas supersaturation at one of three depths, 10, 50, or 100 cm (±10 cm volitional range), the fish died most quickly in the shallow cage and most slowly in the deep cage (Fig. 1A). Dose-response curves ($Y = aX^b$, where $Y$ is median survival time, $X$ is excess dissolved gas pressure, and $a$ and $b$ are constants) yielded $r^2$ values of 0.99, 0.96, and 0.83 for the 10, 50, and 100 cm depths, respectively.

When the median survival times were plotted against depth-corrected excess gas pressures, a single curve could be fitted to the data yielding an $r^2$ value of 0.95. Because the fish could have selected depths slightly above or below the midcage depths of 10, 50, and 100 cm used in the previous calculations, we attempted to fit better curves for other possible exposure depths. Best fits were obtained when fish in the shallow cage were assumed to have been near the bottom of their cage and those in the other two cages near the top. Figure 1B represents the response of fish at assumed depths of 17, 46, and 93 cm and has an $r^2$ value of 0.97. The results support the concept that depth provides protection against the lethal effects of supersaturation in

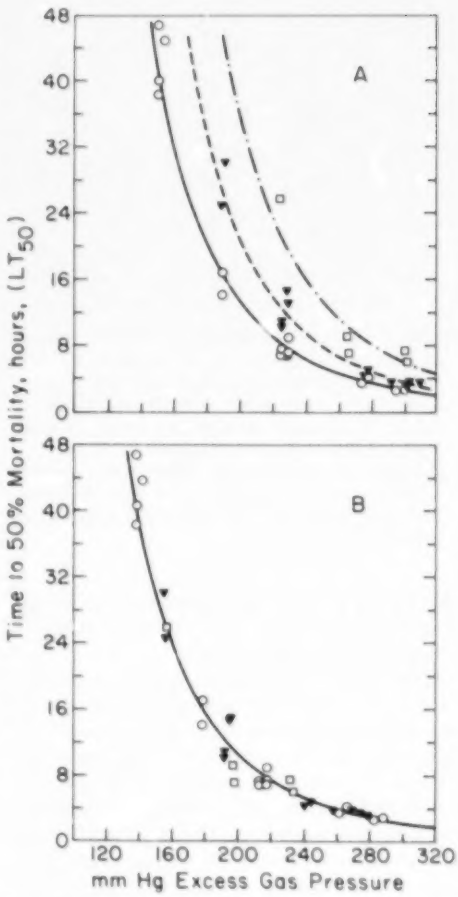


FIGURE 1.—*Relationship* ($Y = aX^b$) *between time to 50% mortality* (Y) *and mm Hg excess total gas pressure* (X) *for steelheads held at water depths of 10 cm (circles), 50 cm (triangles), and 100 cm (squares). Curves in A, calculated without allowance for hydrostatic pressure, have constants* (a, b) *of (1.243, −3.944), (4.468, −3.761), and (1.892, −3.187) for 10, 50, and 100 cm, respectively. The curve in B, calculated after allowance for hydrostatic pressure, has constants* (a, b) *of (4.830, −3.768).*

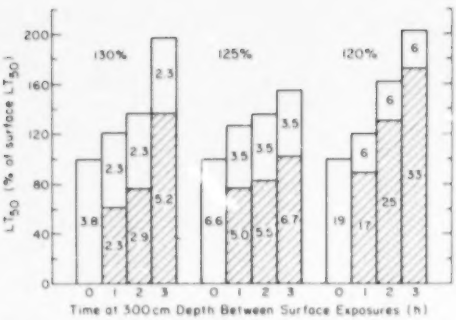


FIGURE 2.—*Time (at the water surface) to 50% mortality for steelheads allowed 0, 1, 2, or 3 hours at 3-m depth between shallow-water exposures to 120, 125, or 130% air saturation. White portions of histograms represent initial shallow exposures, crosshatched portions the second shallow exposures. Hours of exposure are indicated in the histograms; the single or lower number in each bar is time to 50% mortality for single or second surface exposures, respectively. Values for 120 and 125% saturation are means of two tests.*

direct proportion to the reduction in excess gas pressure provided by the hydrostatic pressure of the overlying column of water.

Hydrostatic pressure thus appears to provide a calculable protection against the apparent excess gas pressure measured at the water surface. The question remained as to the effectiveness of hydrostatic pressure in reversing or retarding the effects of a prior acute exposure to excess gas pressure. Consequently, we exposed fish to several levels of excess gas pressure near the surface for a period determined to be just short of the time required to produce death in the most susceptible fish. The fish were then lowered to a depth (3 m) calculated to produce total compensation. After 1, 2, or 3 hours, the fish were returned to the surface and their response observed; time to 50% mortality was the index of effect.

The simplest mechanism for enhanced survival of fish lowered to compensatory depths is resolubilization of already formed gas emboli. Among other effects, this would allow renewed blood flow to ischemic tissues, reoxygenation of tissue, and clearance of metabolic waste products.

Comparison of LT50's of fish kept near the surface with those of fish given shallow reexposure following depth compensation is complicated by several factors. These include gas equilibration time of body fluids (Beyer et al. 1976); unknown relationships among degree of equilbration, internal gas pressure, and onset and rate of emboli formation; and the relative rates of emboli formation in fish held at the surface and those brought into shallow water from a compensatory depth.

Our data suggest that for steelheads first exposed to excess surface gas tensions for nonlethal periods, the minimum time for full compensation at a 3-m depth is 2–3 hours at 125 and 130% saturation and 1–2 hours at 120% saturation (Fig. 2). However, at all three levels of air supersaturation, the longer fish remained at depth, the greater their subsequent tolerance of shallow conditions (as indicated by their higher LT50's).

Active fish are reported to be more susceptible to gas bubble disease than quiescent ones (Stroud and Nebeker 1976). Our handling of fish during the depth-compensation experiments could have induced varying activity levels and caused some of the differences noted in LT50 values. Still, our results suggest that physiological adaptation, as well as hydromechanical compensation, may occur in fish allowed a deep refuge in air-supersaturated water.

## Acknowledgments

We thank Robert Trippel and Donald Stevens for design and construction of the test apparatus. Donald Pierce and James Andros provided statistical assistance and Alan Nebeker provided technical consultation.

## References

BEYER, D. L., B. G. D'AOUST, AND L. S. SMITH. 1976. Decompression-induced bubble formation in salmonids: comparison to gas bubble disease. Undersea Biomedical Research 3:321–338.

DAWLEY, E. M., M. SCHIEWE, AND B. MONK. 1976. Effects of long-term exposure to supersaturation of dissolved atmospheric gases on juvenile chinook salmon and steelhead trout in deep and shallow test tanks. Pages 1–10 *in* D. H. Fickeisen and M. J. Schneider (1976).

FICKEISEN, D. H., AND J. C. MONTGOMERY. 1978. Tolerances of fishes to dissolved gas supersaturation in deep tank bioassays. Transactions of the American Fisheries Society 107:376–381.

FICKEISEN, D. H., AND M. J. SCHNEIDER, editors. 1976. Gas bubble disease. CONF-741033, Technical Information Center, Energy Research and Development Administration, Oak Ridge, Tennessee, USA.

FICKEISEN, D. H., M. J. SCHNEIDER, AND J. C. MONTGOMERY. 1975. A comparative evaluation of the Weiss saturometer. Transactions of the American Fisheries Society 104:816–820.

NEBEKER, A. V., D. G. STEVENS, AND J. R. BRETT. 1976. Effects of gas supersaturated water on freshwater aquatic invertebrates. Pages 51–65 *in* D. H. Fickeisen and M. J. Schneider (1976).

RUCKER, R. 1972. Gas bubble disease of salmonids: a critical review. United States Bureau of Sports Fisheries and Wildlife Technical Paper 58.

SAMUELSON, D. F. 1976. Water quality: Western Fish Toxicology Station and western Oregon rivers. United States Environmental Protection Agency, National Environmental Research Center, Ecological Research Series EPA-600/3-76-077.

STROUD, R. K., G. R. BOUCK, AND A. V. NEBEKER. 1975. Pathology of acute and chronic exposure of salmonids to supersaturated water. Pages 435–449 *in* Chemistry and physics of aqueous gas solutions. The Electrochemical Society, Princeton, New Jersey, USA.

STROUD, R. K., AND A. V. NEBEKER. 1976. A study of the pathogenesis of gas bubble disease in steelhead trout (*Salmo gairdneri*). Pages 66–71 *in* D. H. Fickeisen and M. J.Schneider (1976).

WEITKAMP, D. E. 1976. Dissolved gas supersaturation: live cage bioassays at Rock Island Dam, Washington. Pages 24–36 *in* D. H. Fickeisen and M. J. Schneider (1976).

WEITKAMP, D. F., AND M. KATZ. 1980. A review of dissolved gas supersaturation literature. Transactions of the American Fisheries Society 109:659–702.

WOLKE, R. E., G. R. BOUCK, AND R. K. STROUD. 1974. Gas bubble disease: a review in relation to modern energy production. Pages 239–266 *in* S. B. Saila, editor. Fisheries and energy production: a symposium. Lexington Books, Lexington, Massachusetts, USA.